

SUMMARY

Milk stasis, blocked ducts, inflammatory or infectious mastitis, and breast abscess represent the spectrum of maternal hyperlactation syndrome. Management includes decreasing the rate of milk synthesis, improving milk removal out of the breast, and antibiotic therapy for ascending lactiferous duct infections and mastitis. Thriving infants who choke and splutter at the breast, feed frequently, are colicky, and have explosive, watery bowel movements have infant hyperlactation syndrome and are managed by decreasing quantity and increasing quality of breast milk drunk.

RÉSUMÉ

La stase laiteuse, le blocage des canaux galactophores, la mastite inflammatoire ou infectieuse et l'abcès du sein forment le spectre du syndrome d'hyperlactation maternelle. Le traitement comprend notamment la réduction de la synthèse du lait, l'amélioration des méthodes utilisées pour extraire le lait du sein et l'antibiothérapie pour traiter l'infection ascendante des canaux lactifères et la mastite. Les nourrissons qui progressent bien mais qui toussent et s'étouffent pendant l'allaitement, qui présentent des coliques et dont les selles sont liquides et explosives souffrent du syndrome d'hyperlactation infantile. Le traitement consiste à réduire la quantité de lait maternel absorbé et à améliorer la qualité.

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Too much of a good thing

Maternal and infant hyperlactation syndromes

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HEALTH PROFESSIONALS AND FAMILIES know that breastfeeding is the best way to feed babies. Across Canada, the number of mothers who choose to breastfeed is increasing, and mothers are consulting their family physicians more frequently, seeking answers to their breastfeeding difficulties.

During the last decade, while I consulted with more than 8000 breastfeeding families, characteristic clustering of symptoms and signs gradually became apparent. I have coined the terms maternal and infant hyperlactation syndromes to describe two such clusters. This article describes these syndromes, explains their pathogenesis, and outlines management plans that clinical experience has shown to be effective.

Maternal hyperlactation syndrome

Milk stasis, blocked ducts, inflammatory mastitis, infectious mastitis, and

breast abscess are all common and are part of a spectrum of maternal hyperlactation syndrome (*Figure 1*).

The rate of breast milk synthesis varies from mother to mother and depends on a variety of rate-controlling factors. In my clinical practice, approximately 15% of mothers have a high rate of milk synthesis (60 mL/h or more), and 15% of mothers have a low rate of synthesis (10 mL/h or less).

In a lactating breast, blocked ducts, milk stasis, mastitis, and abscess formation are all consequences of residual milk due to incomplete breast drainage, in the same manner as residual urine due to incomplete bladder emptying might result in ascending urinary tract infections and pyelonephritis. In my experience, most mothers experiencing any or all of these symptoms have a high rate of milk synthesis, an abundant milk supply, and thriving infants, or else mothers have started to wean, skipping some feeds, and have not drained their breasts regularly.^{1,2}

Problems also occur when a mother with a rapid rate of milk synthesis switches her infant from one breast to the other before the first side has been adequately drained. The situation is

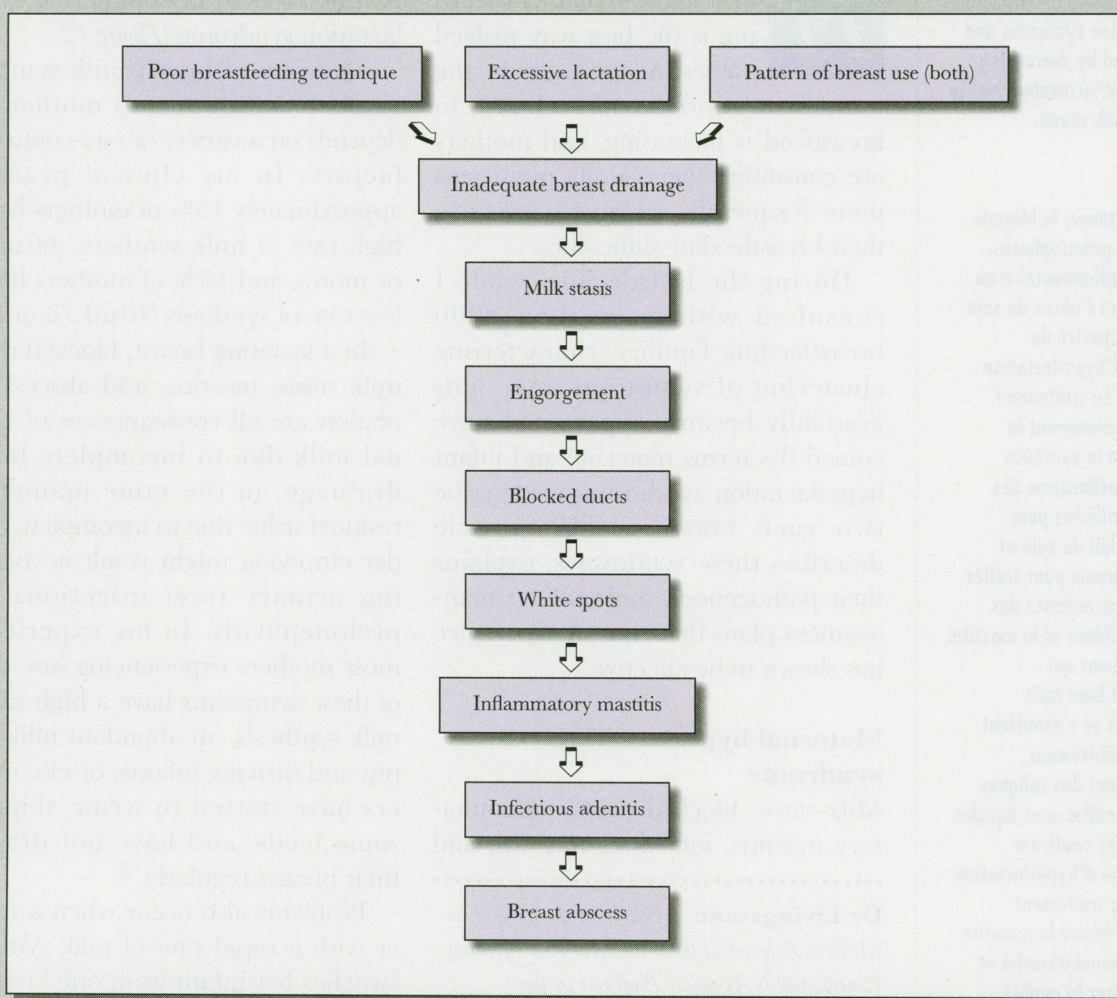
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aggravated by poor positioning and latching the infant on the breast. The infant is unable to grasp the breast and suckle effectively. The milk-ejection reflex causes a rapid let-down of milk and the baby quickly becomes satiated, but might not have emptied all of the lactiferous ducts. If this happens repeatedly, some of the ducts and lobules constantly remain full, leading to outlet obstruction, poor drainage, blocked ducts, and milk stasis. Mothers might complain of "knifelike" cramps or "shooting" pains deep in the breast, often between feeds. A firm, lumpy, slightly tender quadrant in the breast might be felt. Over

time, if this area is not drained, it becomes inflamed and erythematous, signifying an inflammatory mastitis. The high rate of milk synthesis combined with continually poor drainage of a segment of the breast results in the stagnant milk becoming secondarily infected with common skin pathogens via an ascending lactiferous duct infection and leads to infectious mastitis.

Poor positioning might lead to tongue friction on the nipple causing a break in the skin. Fifty percent to 60% of sore, cracked nipples become contaminated with *Staphylococcus aureus*.³ A superficial wound infection can also develop into cellulitis that

Figure 1. Maternal hyperlactation syndrome



might subsequently spread into the deeper, glandular tissue, causing mastitis. Mastitis might also be caused by a blood-borne infection; this is uncommon but more likely in nonpuerperal mastitis. Postpartum endemic mastitis occurs in approximately 1% to 5% of mothers.

Inadequately treated mastitis can develop into a breast abscess. A high fluctuating fever with chills and general malaise, associated with a firm, well-demarcated, tender, fluctuating mass (usually with erythema of the skin) indicates abscess formation, but in some rare instances systemic symptoms are absent. Needle aspiration under local anesthetic using an 18-gauge needle or ultrasonography of the breast are useful diagnostic techniques to identify a collection of fluid or pus and distinguish it from mastitis or a galactocele.

Breastfeeding management goals

Improve drainage. The treatment of choice is to improve milk removal and drainage of each breast. Infants are usually effective at transferring out the milk and draining each segment if they are positioned and latched correctly. The modified

cradle position (*Figure 2A*) allows the mother to cup the affected breast with her hand and apply firm pressure over the outer quadrant and massage the milk toward the nipple while the infant suckles. If the milk is flowing rapidly, the mother should stop massaging the breast. Switching breastfeeding position and using the under-the-arm hold (*Figure 2B*) improves drainage of the inner quadrants of the breast.⁴

Breastfeeding should start on the fullest breast, and the baby should remain on this breast until all areas feel soft. As the pressure in the duct is relieved, breast pain and discomfort lessens. A small white dot on the nipple might become visible, indicating a blocked nipple pore and outlet obstruction. A sterile needle can be used to lift the epithelium skin off the nipple pore to release the milk. On firm massage, a thick stream of milk will often gush out, indicating patency (*Figure 3*).

On occasion, the inspissated milk is thickened and breastfeeding is ineffective at removing the milk. Manual expression or using a good breast pump might help. Piston-action hand pumps are cheap and easy to clean. Battery-operated pumps

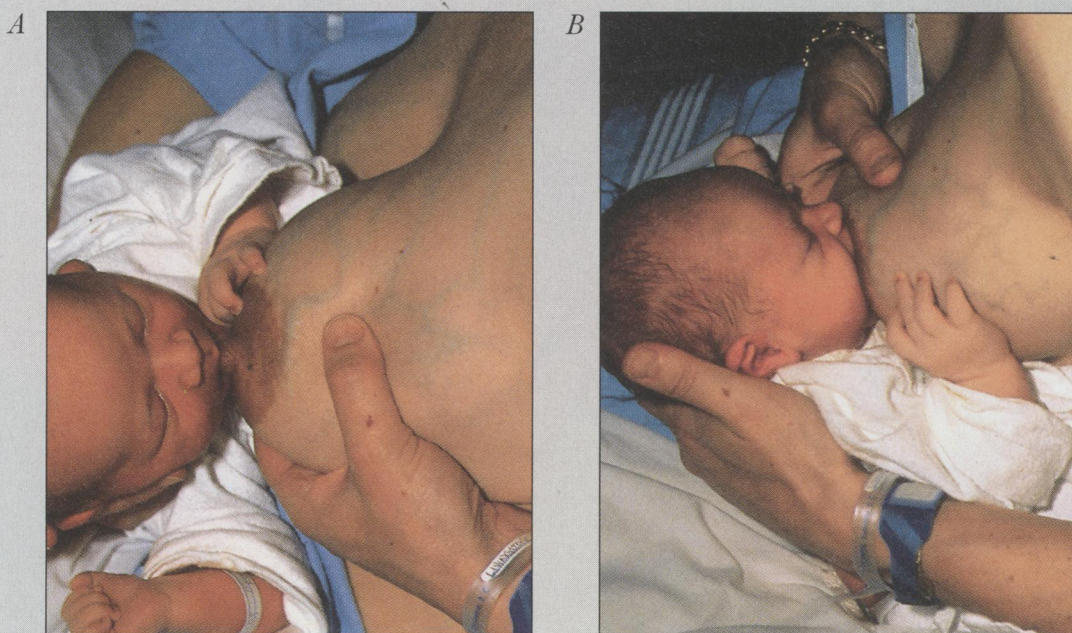


Figure 2. Correct breastfeeding positions: A) Modified cradle position, B) Under the arm position.

are expensive and not always adequate. Large, hospital type, electrical breast pumps are recommended, if available. Mothers should be shown how to massage their breasts firmly toward the nipple while pumping, in order to dislodge the milk.

If breast expression fails to relieve the engorged segment, a technique known as manual stripping might be used.^{5,6} This involves cupping the breast, applying firm, steady pressure over the tender section, starting from the periphery over the rib cage, and drawing the fingers and thumb slowly together toward the nipple, stripping out thickened milk or pus. Repeat the procedure several times. The skin must be well lubricated before attempting to do this. Analgesia might be necessary, but even with mastitis, the discomfort lessens as the procedure continues. The intraductal pressure is relieved as milk or pus is slowly extruded. Mothers must be taught how to use this technique themselves, by standing in the shower, using soapy fingers, every few hours until the breast feels softer.

If a breast abscess has formed, incision and drainage under local or general anesthesia is required.⁵ Repeat needle aspiration has been

tried, but is usually inadequate. The incision should be radial, not circumferential, to minimize duct severance. A large drain should be inserted and daily irrigations continued until the cavity closes. It is important that dressings be applied in such a manner that the baby can continue to breastfeed, or else the mother should use an efficient breast pump. Regular drainage prevents further milk stasis and maintains lactation (*Table 1*).

Treat infection. Correct breastfeeding technique and improved drainage of milk are the sine qua non of treatment, but antibiotic therapy might be necessary. Inflammatory mastitis occurs within 12 to 24 hours of milk blockage, leading to infectious mastitis within 24 to 48 hours.

Under normal conditions, the milk leukocyte count is $< 10^6/\text{mL}$ of milk and the bacterial count is $< 10^3$ bacteria/mL. Within 48 hours of breast symptoms, the leukocyte count increases to $> 10^6/\text{mL}$ of milk, but the bacterial count remains low. This is considered non-infectious inflammation of the breast, and improved drainage of milk will resolve the situation quickly.



Figure 3. Opening a "white dot"

Infectious mastitis is defined as having a bacterial count higher than 10^6 /mL of milk.^{7,8} In this situation the addition of antibiotics would be recommended, but in clinical practice, treatment is usually empirically started. Common bacterial pathogens include *S aureus*, *Escherichia coli*, β -hemolytic *Streptococcus* Group A with occasional *Streptococcus faecalis*, and *Klebsiella pneumoniae*. In contrast, nonpuerperal breast infections are mixed infections with a large anaerobic component.⁹ Antibiotics of choice include penicillinase-resistant penicillins, such as dicloxacillin or erythromycin, cephalosporins, sulfonamides, and clindamycin. A 10-day course is required. Minimal amounts of these antibiotics are excreted in breast milk, and it is considered safe to continue to breastfeed.¹⁰

Staphylococcus toxins might, in rare circumstances, be ingested by infants, but continuation of breastfeeding should always be recommended.¹¹ Abrupt weaning could lead to increased milk stasis and abscess formation. If a mother chooses to wean abruptly, bromocriptine (2.5 mg twice daily for 14 days) may be used with caution; side effects, including nausea, headache, and postural hypotension, might be experienced.

Clinical improvement is usually seen within 24 to 48 hours; the erythema subsides, the fever decreases, and breast pain improves. A persistent mass that fluctuates might indicate abscess formation.

Decrease rate of milk synthesis. Maternal hyperlactation syndrome can be prevented by decreasing the rate of milk synthesis and improving milk removal and breast drainage by correct breastfeeding technique. The rate of milk synthesis is controlled by central and local factors.¹² Direct breast stimulation from infant suckling

causes a twofold or threefold rise in prolactin and releases other pituitary lactogenic hormones. These lactogens trigger milk production by the mammary acinar glands.

Frequent breast stimulation facilitates increased milk synthesis. A 20-minute period of breast stimulation causes peak prolactin surges that take about 2 hours to decline to baseline levels. Milk synthesis occurs during this period. Decreasing the amount of breast stimulation by

decreasing the frequency and duration of breastfeeding lowers prolactin levels and reduces the rate of milk synthesis. As milk pools in the lactiferous ducts, a local inhibitor peptide collects within the breast and decreases ongoing milk production via a local negative feedback mechanism.¹³⁻¹⁵ Therefore, decreasing the frequency of breast drainage and delaying the removal of inhibitor peptides results in a decrease in the rate of milk synthesis (Figure 4).

In practical terms, encouraging the infant to remain at one breast per feed until he or she is full and spontaneously releases the breast and using both the football and modified cradle positions allows thorough drainage of all segments and

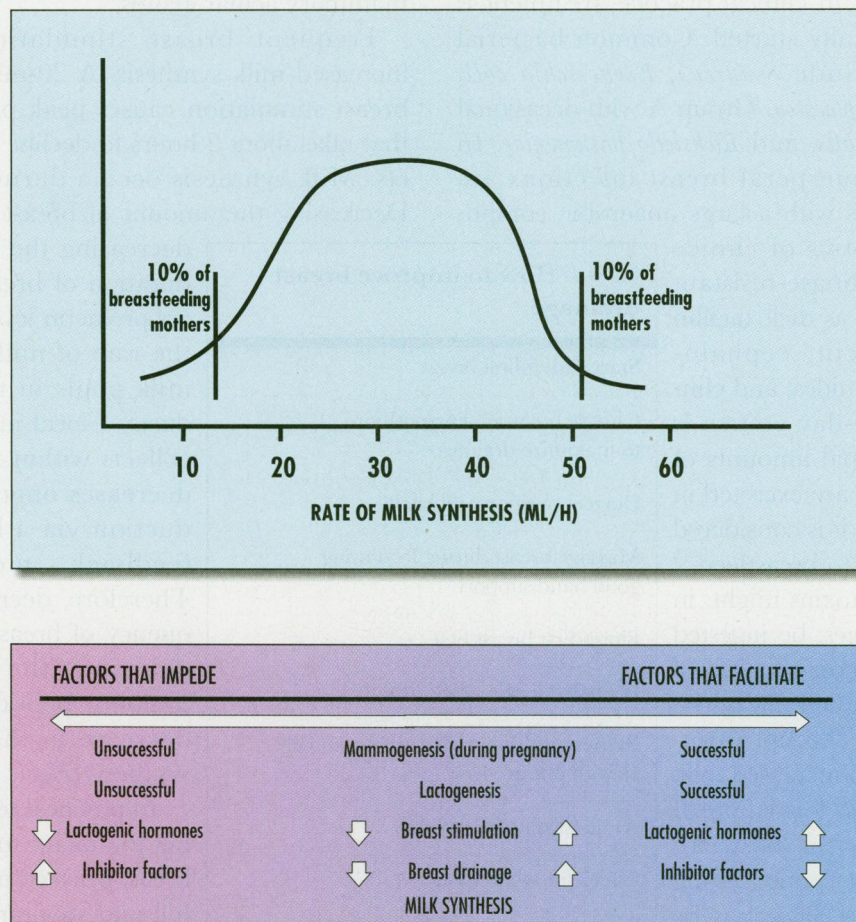
prevents milk stasis. The quantity of milk drunk is less, but the fat content and calorific value increases as the feed progresses. A higher fat intake often satiates the infant for a longer period and decreases the hunger drive.¹⁶ The interval between feeds is lengthened, and milk synthesis declines. The second breast remains full for a longer period, resulting in a local buildup of inhibitor that reduces milk synthesis in that breast.

Prevent recurrence. Milk stasis can be prevented by correct breastfeeding technique to ensure a proper latch and by regular drainage and avoiding

Table 1. How to improve breast drainage

Start with fullest breast
Choose breastfeeding position to maximize drainage
Correct position and latch
Massage breast during feed using good hand support
Finish first breast first
Let baby decide when to stop
Remove residual milk either manually or with pump
Avoid missing breastfeeds
Check for white spots
Apply warm compress or shower
Facilitate the milk ejection reflex
Avoid nipple shields

Figure 4. Hypothetical model of changes in rate of maternal milk synthesis



skipping feeds. Sleeping through the night; returning to work; introduction of breast milk substitutes, such as bottles of formula; introduction of table foods; and weaning are all typical periods when breastfeeds might be missed. The resultant "breast confusion" can lead to inadequate drainage and subsequent infections.¹⁷

Mothers with abundant milk should become skilled at palpating their breasts and checking for lumpiness, especially after each feed. They should remove their bras before feeding if practical. Areas of breast lumpiness that persist after breastfeeding might indicate milk stasis or a blocked duct. After feeds, thorough expression of this residual milk should relieve the situation.

Gradual baby-led weaning is best; abrupt weaning might predispose mothers with high milk production to develop maternal hyperlactation syndrome.

Offer supportive measures. Parenting is tiring for mothers. Mastitis, an inflammatory process, can be complicated by infection and produce systemic symptoms in an already exhausted mother. Additional home help is mandatory; bed rest is advisable; analgesia, such as acetaminophen alone or combined with codeine, might be necessary. Hot compresses to the breast, before breastfeeding or milk expression, encourage blood flow and smooth muscle relaxation,

which help milk transfer. Cold compresses after feeds might decrease inflammation and edema.

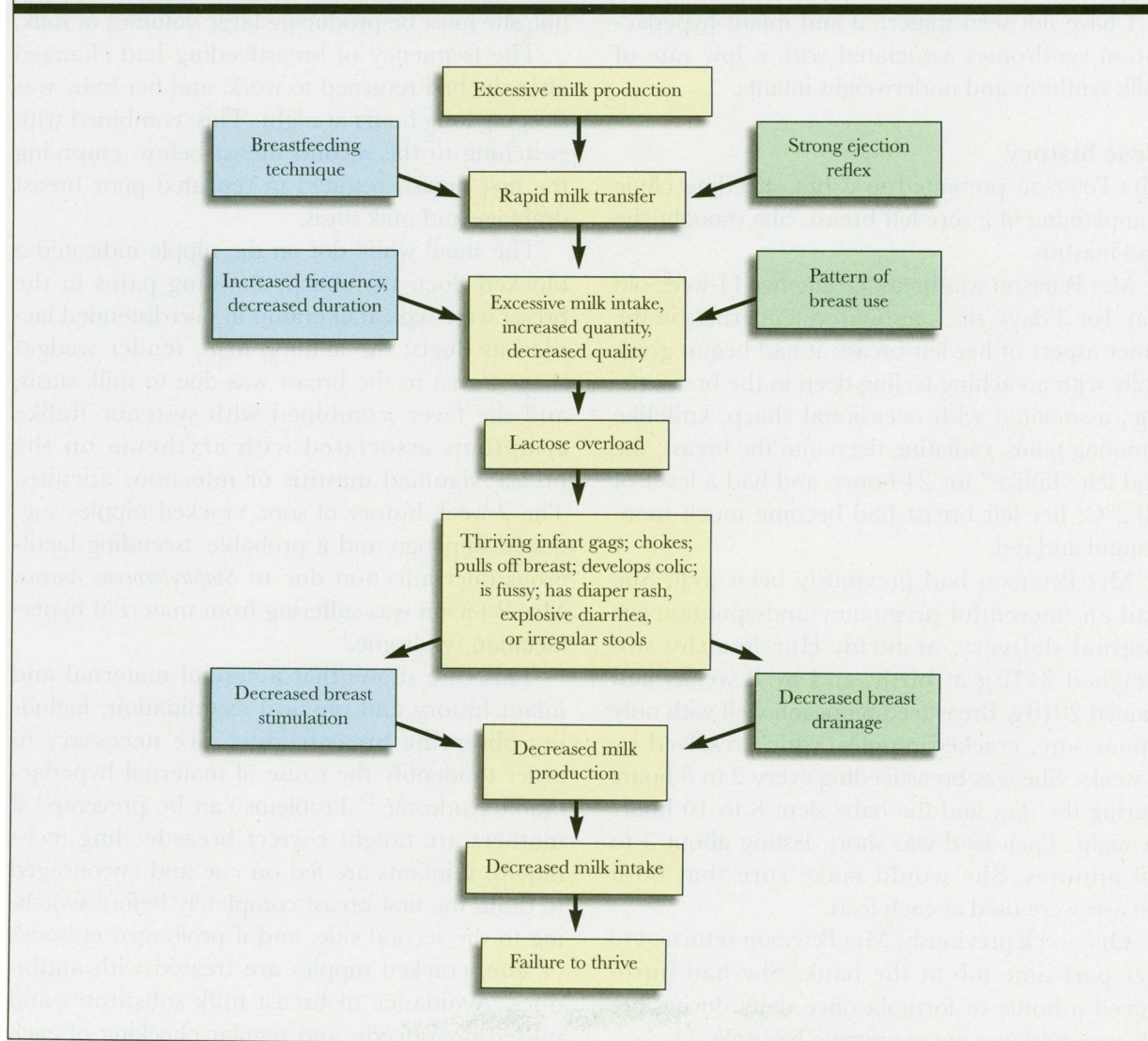
Infant hyperlactation syndrome

In my clinical practice, the infants of mothers with maternal hyperlactation syndrome often suffer from their own characteristic cluster of symptoms and signs, which I have termed infant hyperlactation syndrome. The infants are usually thriving, constantly demanding to be fed, yet they choke and sputter during feeds because of the strong milk ejection reflex. They pull away from the breast after suckling for only a few minutes

and are gassy, fussy, and colicky. Many have frequent watery diarrhea and occasional diaper rash.

These infants drink a large quantity of milk quickly, sometimes as much as 120 to 140 mL of milk within 5 or 10 minutes. This rapid ingestion of lactose-rich milk might cause symptoms of lactose overload because the small amount of lactase present in infants' intestines is unable to fully metabolize all the available lactose. Undigested lactose leads to symptoms typical of lactose intolerance, such as gas, colic, flatulence, and explosive, watery diarrhea that is acidic. After several weeks some infants who initially thrive start to

Figure 5. Infant hyperlactation syndrome



reject the breast because of the unpleasantly strong milk ejection and gradually spend less and less time sucking at the breast but more time sucking their thumbs or pacifiers.¹⁸

The infants start to fail to thrive, and milk production dwindles. These babies can be lured back to the breast if the mother recognizes the problem, if she feeds on a softer breast when the infant is sleepy, burps frequently, and remains on one breast until it is fully drained before switching to the second side. In this way, the infant obtains a better quality feed with a higher fat content and less lactose instead of a larger quantity of milk with more lactose and less fat (Figure 5).

I have not seen maternal and infant hyperlactation syndromes associated with a low rate of milk synthesis and underweight infants.

Case history

Mrs Peterson presented to a breastfeeding clinic complaining of a sore left breast. She thought she had mastitis.

Mrs Peterson was breastfeeding her 14-week-old son. For 3 days, she had noticed tenderness in the inner aspect of her left breast; it had begun gradually with an aching feeling deep in the breast tissue, associated with occasional sharp, knifelike shooting pains, radiating deep into the breast; she had felt "flulike" for 24 hours, and had a fever of 38.2°C; her left breast had become much more painful and red.

Mrs Peterson had previously been well. She had an uneventful pregnancy and spontaneous vaginal delivery, at term. Her healthy son weighed 3470 g at birth, and by 8 weeks had gained 2010 g. Breastfeeding began well with only minor sore, cracked nipples, which resolved by 2 weeks. She was breastfeeding every 2 to 3 hours during the day, and the baby slept 8 to 10 hours at night. Each feed was short, lasting about 5 to 10 minutes. She would make sure that both breasts were used at each feed.

One week previously, Mrs Peterson returned to her part-time job at the bank. She had introduced a bottle of formula once daily during her absence and was not expressing her milk.

On examination, the baby was a large, thriving boy, weighing 7010 g, above the 90th percentile. Mrs Peterson looked tired; her temperature was 39.1°C; her breasts were tense and full of milk. The left inner quadrant was erythematous with a palpable wedge-shaped mass radiating toward the axilla. On close inspection of the left nipple, a small white dot was visible, which coincided with the blocked segment of breast. Manual expression of milk revealed several patent ducts, but milk could not be expressed from the left upper outer quadrant, under the white dot.

After reviewing the history and physical findings, I concluded that, because her infant was gaining 42 g/day and her breasts were brimming full, she must be producing large volumes of milk.

The frequency of breastfeeding had changed after she had returned to work, and her baby was sleeping long hours at night. This, combined with switching to the second breast before emptying the first breast, resulted in repeated poor breast drainage and milk stasis.

The small white dot on the nipple indicated a blocked duct; the sharp, shooting pains in the breast were typical of cramp in overdistended lactiferous ducts; the aching, firm, tender wedge-shaped area in the breast was due to milk stasis, and the fever (combined with systemic flulike symptoms associated with erythema on the breast) signified mastitis or infectious adenitis. The 2-week history of sore, cracked nipples suggested impetigo and a probable ascending lactiferous duct infection due to *Staphylococcus aureus*. Mrs Peterson was suffering from maternal hyperlactation syndrome.¹

This case shows that a careful maternal and infant history and physical examination, including observing breastfeeding, are necessary in order to identify the cause of maternal hyperlactation syndrome.¹⁹ Problems can be prevented if mothers are taught correct breastfeeding techniques, if infants are fed on cue and encouraged to drain the first breast completely before switching to the second side, and if prolonged episodes of sore, cracked nipples are treated with antibiotics. Avoidance of breast milk substitutes and missed breastfeeds, and regular checking of each

breast for early signs of milk stasis, will also help.²⁰ ■

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For further reading

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Antibiotic and β -lactamase inhibitor

Indications: Infections caused by susceptible β -lactamase-producing strains of designated bacteria: upper respiratory tract and skin and soft tissue infections due to *S. aureus*; lower respiratory tract infections due to *H. influenzae*, *K. pneumoniae*, *S. aureus* or *Moraxella* (Branhamella) catarrhalis; otitis media due to *H. influenzae* or *Moraxella* (Branhamella) catarrhalis; urinary tract infections due to *E. coli*, *P. mirabilis* or *Klebsiella* species and sinusitis due to *H. influenzae* or *Moraxella* (Branhamella) catarrhalis. **Contraindications:** History of hypersensitivity to the penicillins, clavams or cephalosporins; history of Clavulin-associated jaundice/hepatic dysfunction; infectious mononucleosis suspected or confirmed. **Warnings:** Before initiating therapy, careful inquiry should be made concerning previous hypersensitivity reactions to penicillin, clavams, cephalosporins or other allergens, as serious and occasionally fatal hypersensitivity (anaphylactoid) reactions have been reported. If an allergic reaction occurs, discontinue Clavulin and initiate appropriate therapy. Serious anaphylactoid reactions require immediate emergency treatment with epinephrine. Oxygen, i.v. steroids and airway management, including intubation, should also be used as indicated. Use with caution in patients with evidence of hepatic dysfunction. Hepatic toxicity associated with the use of Clavulin is usually reversible. On rare occasions, deaths have been reported (less than 1 death reported per estimated 4 million prescriptions worldwide). These have generally been cases associated with serious underlying diseases or concomitant medications. **Precautions:** Periodic assessment of renal, hepatic and hematopoietic function should be made during prolonged therapy. Clavulin is excreted mostly by the kidney. Reduce the dose or extend the dose interval for patients with renal dysfunction in proportion to the degree of loss of renal function. The possibility of superinfection (usually involving *Aerobacter*, *Pseudomonas* or *Candida*) should be kept in mind. If it occurs discontinue Clavulin and institute appropriate therapy. The occurrence of a morbilliform rash following the use of ampicillin in patients with infectious mononucleosis is well documented. This reaction has also been reported following the use of amoxicillin. A similar reaction would be expected with Clavulin. As with all medicines, use in pregnancy is not recommended, especially during the first trimester, unless the anticipated benefit justifies the potential risk to the fetus. Penicillins have been shown to be excreted in human breast milk. It is not known whether clavulanic acid is excreted in breast milk. Caution should be exercised if administered to a nursing mother. In common with other broad spectrum antibiotics, Clavulin may reduce the efficacy of oral contraceptives and patients should therefore be advised accordingly. **Adverse Reactions:** Gastrointestinal: Nausea, vomiting, diarrhea, abdominal cramps, flatulence, constipation, anorexia, colic pain, acid stomach, intestinal candidiasis and pseudomembranous colitis. If gastrointestinal reactions are evident, they may be reduced by taking Clavulin at the start of the meal. The incidence of gastrointestinal side effects tends to be proportional to dose and tends to be greater in children than adults. **Hypersensitivity Reactions:** Erythematous maculopapular rash, urticaria, anaphylaxis and pruritis. A morbilliform rash in patients with mononucleosis. Rarely erythema multiforme and Stevens-Johnson syndrome have been reported. Other reactions including angioedema, toxic epidermal necrolysis and exfoliative dermatitis, as in the case of other β -lactam antibiotics, have been seen rarely. **Interstitial nephritis (rarely):** Liver: Transient hepatitis and cholestatic jaundice have been reported rarely. These events have been noted with other penicillins and cephalosporins. Hepatic events associated with Clavulin may be severe, and occur predominantly in adult and elderly patients. Signs and symptoms usually occur during or shortly after treatment, but in some cases may not become apparent until several weeks after treatment has ceased. Hepatic events are usually reversible, however, in extremely rare circumstances, deaths have been reported. These have almost always been cases associated with serious underlying disease or concomitant medications. Moderate rises in SGOT, alkaline phosphatase and lactic dehydrogenase, and SGPT have been noted in patients treated with ampicillin class antibiotics. The significance of these findings is unknown. **Hemic and Lymphatic Systems:** As with other β -lactams, anemia, hemolytic anemia, thrombocytopenia, thrombocytopenic purpura, eosinophilia, leukopenia, lymphocytopenia, basophilia, slight increase in platelets, neutropenia and agranulocytosis have been reported rarely during therapy with the penicillins. These reactions are usually reversible on discontinuation of therapy and are believed to be hypersensitivity phenomena. Prolongation of bleeding time and prothrombin time (rarely). Other: Vaginitis, headache, bad taste, dizziness, malaise, glossitis, black hairy tongue and stomatitis. **Dosage and Administration:** The absorption of Clavulin is optimized when taken at the start of a meal. **Adults:** For urinary tract, upper respiratory tract, skin and soft tissue infections which are mild to moderate, one Clavulin-250 tablet every 8 hours. For severe infections and lower respiratory tract infections, one Clavulin-500F tablet every 8 hours. **Children:** For urinary tract, upper respiratory tract, skin and soft tissue infections which are mild to moderate, 25 mg/kg/day of Clavulin in equally divided doses every 8 hours. For severe infections, otitis media, sinusitis or lower respiratory tract infections, 50 mg/kg/day of Clavulin in equally divided doses every 8 hours. Children's dosage should not exceed that recommended for adults. Children weighing more than 38 kg should be dosed according to the adult recommendations. Treatment should continue for 48-72 hours beyond the time the patient becomes asymptomatic or bacterial eradication is obtained. At least 10-days' treatment is recommended for infections caused by β -hemolytic streptococci to prevent acute rheumatic fever or glomerulonephritis.

N.B. DO NOT SUBSTITUTE 2 X 250 TABLETS FOR 1 X 500F TABLET. RATIO OF AMOXICILLIN TO CLAVULANIC ACID IS DIFFERENT.

Supplied: Clavulin-250 tablets (250 mg amoxicillin, 125 mg clavulanic acid) in bottles of 100; Clavulin 500F tablets (500 mg amoxicillin, 125 mg clavulanic acid) in bottles of 30, 100. Clavulin-125F Oral suspension (125 mg amoxicillin, 31.25 mg clavulanic acid per 5 ml) and Clavulin-250F Oral suspension (250 mg amoxicillin, 62.5 mg clavulanic acid per 5 ml) in bottles of 100, 150 ml. Product monograph available on request.

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